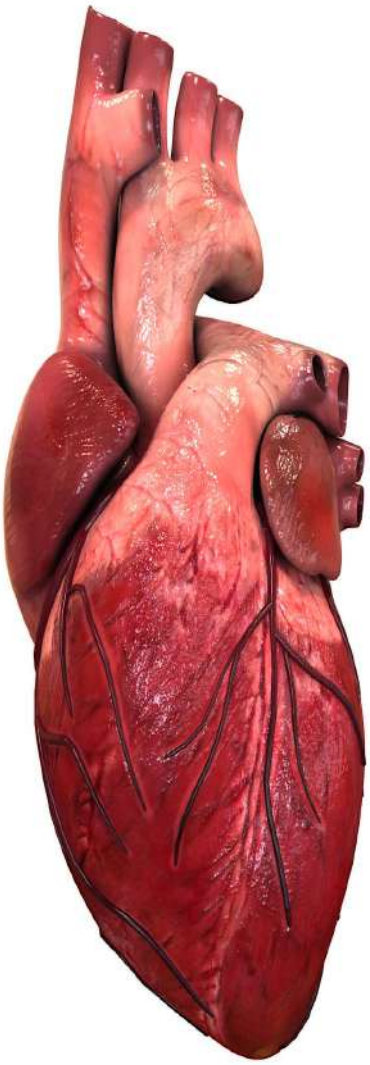
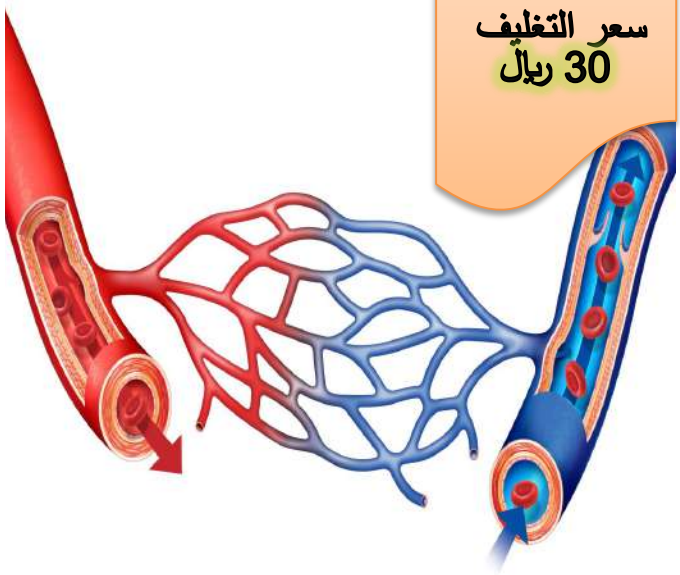
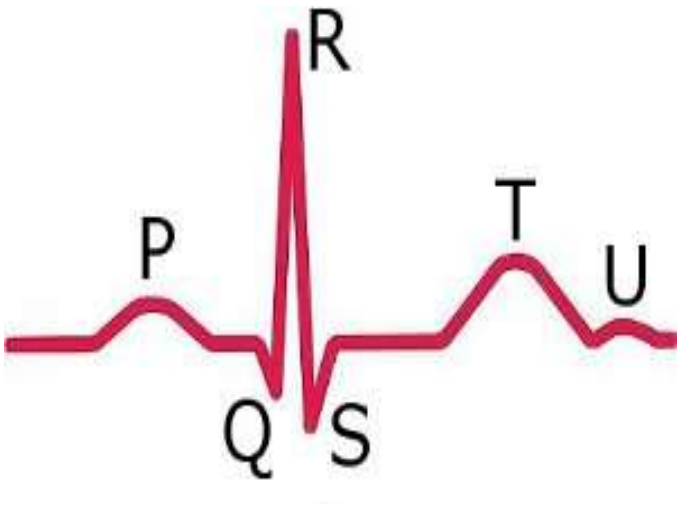
CVS

السعر :
طبعة ملونة
200 ريال
طبعة عادية
165 ريال
سعر التغليف
30 ريال



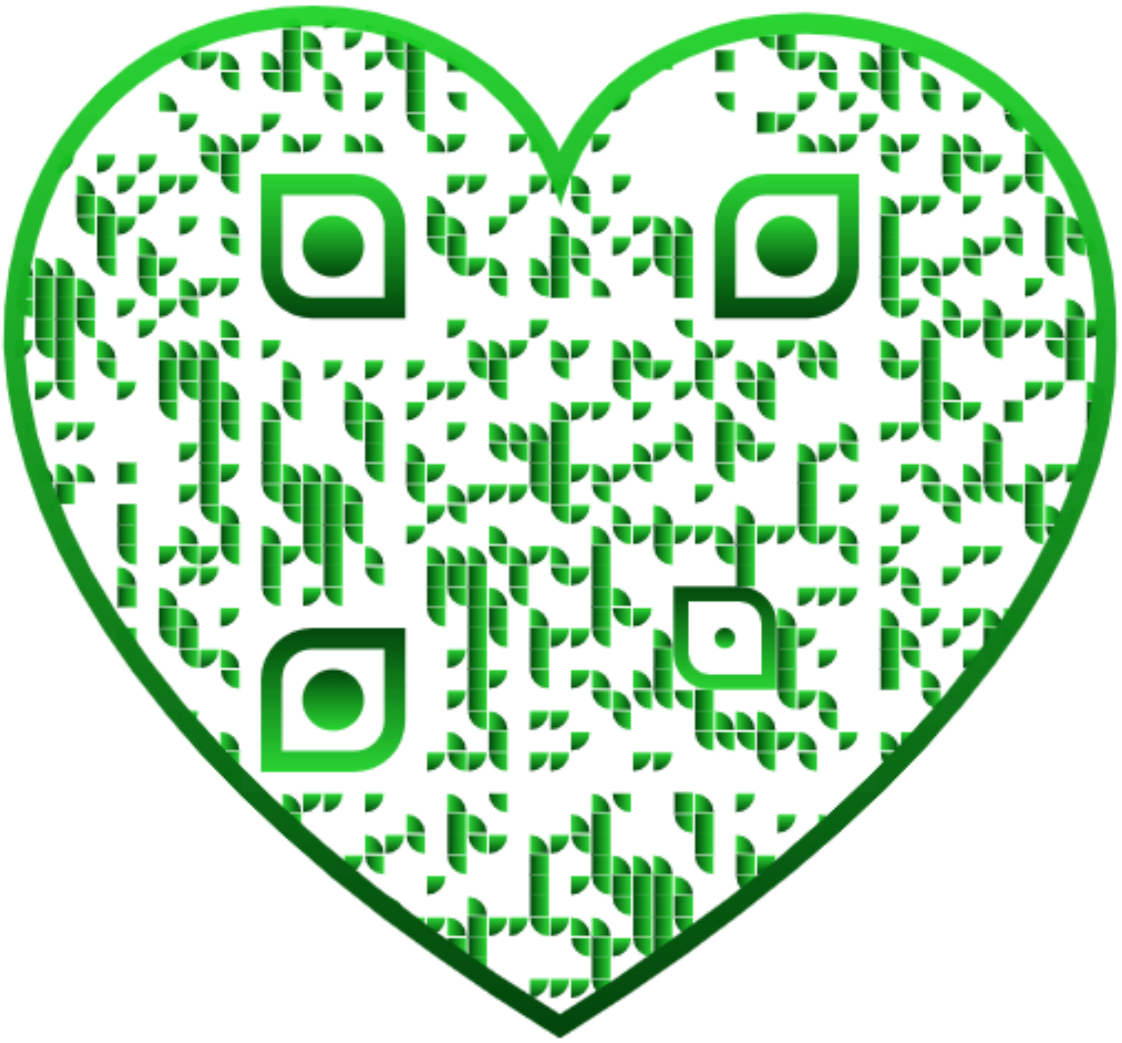




ملخص شرح الدكتور / صادق عبد المغني

هذا الملخص إذا لم يكن يحتوي على جميع كلام الدكتور فهو يحوي أغلبه، ونرجو المعذرة منكم إذا وجدت أي أخطاء إملائية وغيرها من الاخطاء الغير مقصودة،،،

Ebrahim Shaddad



معاني المختصرات في الملخص

P	**Pressure**
ABP	**Arterial Blood Pressure**
VR	**Venous Return**
BVs	**Blood Vessels**
BV	**Blood Volume**
PR	**Peripheral Resistance**
CO	**Cardiac Output**
	Increase
	Decrease
	Leads to
V.D	**Vasodilation**
V.C	**Vasoconstriction**
BF	**Blood Flow**
ANP	**Atrial Natriuretic polypeptide**
AVn	**Atrioventricular node**
SAn	**Sinoatrial node**
-ve	**Negative**
+ve	**Positive**
MI	**Myocardial Infarction**
NS	**Nervous System**
RAB	**Right Atrial Pressure**
SK. M	**Skeletal Muscle**

في حالة وجود أخطاء في الملخص الرجاء منكم تبليغي بارسل موقع الخطاء (رقم الصفحة) و التصحيح للخطاء ، أو أي استفسار على حساب التليقرام

@EbrahimShaddad

لتنزيل الملخصات بشكل PDF من قناة

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CVS

Introduction

- CVS consist of :
  1- Heart 2- Blood vessels
  ★ it is closed system which the blood circulate through it.

- Heart:
  it's a muscular organ consist of myocardial cells.

myocardial cells are dedived into :

- Right myocardial cells
  - Right atrial myocardial cells
  - Right ventricular myocardial cells
- Left myocardial cells
  - Left atrial myocardial cells
  - Left ventricular myocardial cells

★ The tickness of ventricular myocardial cells is more than atrial myocardial cells
The left ventricular myocardial cell are 3 times thicker than right ventricular myocardial cells ... why?

- Because the pressure in the left ventricle is about 80 -120 mmHg
  will right ventricle about 25 mmHg.

- Valves:

There are two types of valves

(A- A-V "Atrioventricular valves:

Are located between atria and ventricles in which they devided into

Right Tricuspid Valve.

Left Mitral Valve.

(B- Semilunar valves:

Are located between the ventricles and the Large blood vessels that arised from them

Aortic valve

Pulmonary Valve

*Function of valves:

Allow passage of blood in one direction

- The Mitral and aortic valves are the most important valves because

The pressure in left ventricle is high.

- Defect of valves mainly due to bacterial infection or inflammation lead to:

1- Stenosis of valves " Narrowing of valves "

or 2- Incompetent or open valves.

- Circulations:

There are three types of circulation:

A- General circulation "Greater circulation".
B- Pulmonary " "Lesser circulation"
C- Special "

(A) General or greater circulation:

Starts from left ventricle that contain oxygenated blood → Aorta → Large arteries → Small arteries → Arterioles → capillaries → Venules → Veins → superior and inferior Vena cava → Right atrium as deoxygenated blood.

(B) Pulmonary or lesser circulation:

Starts from right ventricle that contain deoxygenated blood → Pulmonary arteries → Lung's pulmonary capillaries oxygenation of blood → 4 pulmonary veins → Left atrium as oxygenated blood.

(C) Special circulation:

Mainly: 1- Cerebral circulation
2- Coronary circulation.

* The aorta is one of the largest arteries in the body which arise from left ventricle.
* The pulmonary artery or trunk is similar to any artery in the body with one exception, it contain deoxygenated blood, it is one of the largest arteries in the body.

- Pulmonary veins are similar to all veins in the body except they contain oxygenated blood.

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* Characteristics of Blood vessels:-

* Arteries:-

1- Large arteries:-

Ex:- Aorta
- Pulmonary arteries.
- Other large arteries.

- Have elastic property "Elasticity"

Ejection of blood from the left ventricle to Aorta → Distension of aorta for recive of high blood volum as well as expose of arterial blood pressure.

Elasticity of aorta is very important to maintain of diastolic pressure. "Windkessel effect"

2- Medium and small arteries:-

- They are low muscular resistance.
- Function is to delivery of blood to tissue.

3- Arterioles:

- High muscular resistance BVs.
- Functions: 1- Maintain of ABP by PR
  2- Regulation of blood flow to tissue

$BP = CO \times PR$

- PR depend mainly on diameter of blood vessels

* V.C or V.D of blood vessels during regulation of ABP occur mainly in the arterioles.

- V.D of arterioles → ↓PR → ↓ ABP.
  V.C " " → ↑PR → ↑ ABP.
  as well as
  V.D of arterioles → ↑ BF to tissues.
  V.C " " → ↓ BF " " .

* Capillaries:-
- The smallest type of BVs.
- The only site for exchang between blood and tissue

* Veins:
1- Return the blood to the heart.
2- Storage or reservation of blood
- During rest the highest blood volume located in veins 54 %. of blood.

- Blood volume in BVs:- During rest:

Arteries	Heart	Veins	Capillaries
11 %	12 %	54 %	5 %

Pulmonary vessels = 100 %
18 %.

- The volume or % of blood in arteries 11 %. but with highest pressure.

- As the Pressure appear from aorta 120 mmHg, it reduced gradually toward the vein, in the superior and IVC the BP is about 0-4 mmHg.
- Increase the Pressure in right atrium ↑ may lead to rupture of atrium.

- Functions of the heart:-

A- Function of ventricles:

Ejection of blood

CO ⇒ 5.5 Lit/min time from right ventricle to lungs.

CO ⇒ 5.5 L/min " left " " Aorta.

B- Function of Atria:-

1- Enter way for ventricles.

- The atria collect the blood then deliver it to the ventricles.

2- Secretion of ANP

- ↑ mainly from right atrium, during high BP

Function of ANP → ↑ excretion of Na+ and water in urine → ↓ABP.

3- Contain strech receptors.

↑ VR → ↑ the pressure in the atria → Stimulation of strech receptors → send signals to cardiac center → ↓ABP.

4- Booster pumping action.

- Help or inforce the pumbing of left ventricle.

- During normal state: Weakly function of booster effect.
- During heart failure → Strong function of booster effect occur → inforce the contraction of left ventricle.

* Cardiac Muscles :-

Three taypes of cardiac muscles.

1- Contractile cardiac muscle fibers → 99 %.
2- Nodal conducting fibers
3- Purkinje conducting fibers > 1 %

1- Contractile cardiac muscle fibers:

- Represent about 99 % of cardiac muscle fibers.
- Responsible mainly for contraction, Present in the walls of atria and ventricles.
- Consist of 4 taypes of contractile proteins:
  1- Actin
  2- Myosin
  3- Tropomin "C-T-R"
  4- Tropomyosin.
- Contraction of cardiac muscles called → Systole.
- Relaxation " " " " → Diastole.

2- Nodal conducting fibers:

- Present in the wall of right atrium.
  1- Sainoatrial node "The pace maker of heart"
  2- Atrioventricular node.
- With compare to contractile fibers, they are
  - Smaller
  - Contain few gab Junctions
    ↓ gab junctions → ↓ conduction.

3- Purkinje fibers:-

- Large fibers
  - Contain high number of gab junctions.
    ↑ gab junctions → ↑ conductivity.

★ The lowest area for conduction in heart is:
AV node ... why?
Low no. of gab junctions.

★ Why the SA node is the pacemaker of heart?
- Because it is the main site for initiation and propegation of AP or impulses for heart
- The other muscular organs in the body need nerve supply to generate impulses, but the heart can keep working even if we separate all nerve supply from it " Transplantation "

★ What is the difference between the pacemaker of heart and respiration?
- If the Apneustic center stoped, Person will die. but in the heart the SA node act as the 1ry pacemaker of heart, if SA node stoped the AV node will work as 2ry pacemaker, if AV node stop working, purkinje fiber work as 3ry pacemaker of heart.

* Pathway of initation impulse of heart:

Signals started from SA node → conducted by internode neurons to AV node → AV bundle or bundle of his which it is the only connector between ventricles and atria → To purkinje fiberes → all over the ventricles.

In some people addition bundle appear which it is similar to AV- bunde, Called bundle of kent.

Cause Tachycardia, may lead to special syndrome called "WPW" Waiff-Parkinson-White syndrome.

WPW syndrome cause shortining of P-R interval in ECG → Tachycardia.

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* Fuel of Heart:

- The heart depend mainly on three types of substance as source of energy:

FA - Glucose - Lactate.

A- During rest:

- The heart use mainly FA 60%, Glucose 20% and lactate 20%.

B- During exercise:-

- Heart use Lactate mainly ... why?

Because lactate ↑ in sk. muscles, if it accumulate in sk. muscles → Fatigue or spasm, but the heart use it as source of energy.

* Autonomic innervation to heart:

Sympathetic	Parasympathetic
1- Stimulate of all cardiac properties:	1- inhibition of all cardiac properties:
A- Conductivity.	← A-
B- Contractility.	← B-
C- Autorhythmicity.	← C-
D- Excitability	← D-
2- V.D of coronary arteries	2- V.C of coronary BVs
3- increase of O2 consumption.	3- Decrease the O2 consumption.

NOTES:

* it is very important to know the right and left myocardial cells, espicially during pathology to determine the right or left heart-failure..

* Heart failure is due to:

↑ of ABP → Hypertrophy of myocardial cells → Continuous hypertrophy → Dialated of myocardial cells → Heart failure.

* ↓ Aerobic reaction in absence of O2 → anaerobic reactions → ↑ lactate.

2nd Lecture:-

★ What is the difference between :

- 1ry SA node → The HR = 100 l/min.
- 2ry AV node → " " = 60 l/min.
- 3ry Purkinje fibers → " " = 40 l/min.

- When the Purkinje fiber work, that's mean we are now in complete heart failure or partial heart block.
  So the man can't do exercise or even work.

"Arterial blood pressure" ABP

- Definition
  it is the lateral pressure exerted by blood in the arterial walls.

- How the ABP generated:
  By 1- Pumping of blood, from left ventricle to aorta
  2- PR.
  - we call it peripheral resistance, because it is present in the arterioles and capillaries.
  - PR: Depend on 1- Blood viscosity.
  2- Length of BVs.
  3- Diameter of BVs.

- The normal ABP is about :-
  Systolic Pressure → 90 - 140 mmHg
  Diastolic Pressure → 60 - 90 mmHg

* ↑ or ↓ The diastolic pressure is more dangerous than systolic pressure.

* Function of ABP :-
  1- Maintain of blood flow through different tissues.
  2- Producing of hydrostatic capillary pressure.
  - Hydrostatic pressure :
    It is the pressure in the two ends of capillaries its main function is to filtration of fluid from capillary to tissue it is about 32 mmHg
  - Osmotic pressure :
    Force that cause reabsorption of fluid from tissue to blood, it is about 28 mmHg.
- The hydrostatic pressure depend on ABP :
  ↑ABP → ↑ HP → ↑ Filtration.
  ↓ " → ↓ HP → ↓ Filtration.
- HP is stable 32 mmHg all over the body except kidney 60 mmHg

* Osmotic pressure depend on :
  Plasma proteins
  ↑ plasma protein → ↑ osmotic pressure

3- Specific functions of diastolic P:

A- Maintain blood flow to tissues during diastole

If the diastolic pressure is not present the blood flow to tissues will be intermittent.

B- Any muscle got its blood flow during diastole.

Diastolic P is responsible for BF to coronary arteries → supply heart.

C- Prevent blood stasis "Stagenation" in the arteries during ventricular diastole.

If stasis of blood occur, the blood flow will stop during diastole → need stronger pressure to push the stasis blood → 2-3 minutes cause fatigue of cardiac muscles → no response to impulses → cardiac arrest.

Factors determine and maintain ABP:

1- CO
2- PR
3- Elasticity of aorta
4- Blood volume.

1- CO

- Definition of CO:
  Amount of blood ejected by each ventricle per minute. = 5.5 Lit/min during rest
  = 30-35 Lit/min during exercise
- CO = SV x HR
  Definition of SV:
  Amount of blood ejected by each ventricle per beat.

↑HR → ↑ CO
↑SV → //

2- PR:

- We call it peripheral because it's present in arterioles as well as in capillaries.
- ~~A~~ffected by:
  1- Length of BVs.
  2- Viscosity of blood
  3- Diameter of BVs. → The most important.
  V.C → ↑PR → ↑ ABP.

. Most of the meds for hypertension work on the PR by producing V.D → ↓ABP.

3- Elasticity of large BVs: - Windkessel effect

- During systole the energy produce by contraction of ventricles, this energy will stored in the wall of aorta, Once the blood pass throng the aorta elastic recail of aorta occur by the energy that stored in the wall of aorta.

- Loss of elasticity → 1- ↑ systolic P.
  2- ↓ Diastolic P.

  *Atherosclerosis تصلب الشرايين
  *Arteriosclerosis تصلب الشرايين

- ↑ Elasticity → ↓ ABP.

4- BV:

giving the patient 2 or 3 serum to venous blood
→ ~~↑BV~~ → ↑ ABP.

Physiological factors affect ABP:

1- Age

A- Children:

Low ABP due to high elasticity of BVs.

B- Old age:

High ABP due to sclerosis of BVs → ↓ elasticity.

2- Sex:

A- Female:

Low ABP due to presence of estrogen → ↓ metabolic effect → ↓ cholesterol → ↓ ABP

~~But~~

- Low ABP in female until menopause, because ↓ estrogen → ↑cholesterol → ↑ABP

B- Male:
High ABP

3- Body regions:
- Brachial artery.
- Thigh "Femoral artery" is higher 5-10 mm Hg than normal ABP.
- We use the thigh if the arms are fractured or no upper limbs at all.

4- Body built:
- Obese → High ABP because of high cholesterol level in body
- Thin → low ABP

5- Diurnal variation.
- Morning - weaking up → low ABP
- After thinking or work → High ABP

6- Meal
High ABP because of ↑ Absorption of material into the blood → ↑BV → ↑ABP

• NOTES :

• Headach for one or two months with use of Doxazosin lead to heart failure... why?
You may suffer from hypertension, but the baroreceptors are adapted to the high ABP, so continuous high ABP → Heart failure.

• For 100% true diagnosis of hypertension, we should measure the ABP for 3 days.
The most important pressure is the diastolic p.

• Phenochromocytoma :-
Tumor of adrenal medulla → Continuous secretion of Adrenalin → ↑ ABP.

• Cause of systolic pressure:
Ejection of blood to aorta.

• Cause of diastolic pressure:
Elastic recoil of aorta.

• The difference between systolic and diastolic pressure know as "Pulse Pressure" normally ≤ 40 mm Hg
The people with atherosclerosis or ↓ elasticity of aorta have Hyperdynamic circulation.
e.g pulse pressure is higher than 40 mm Hg.
Hyperdynamic circulation → ↑ systolic p
↓ diastolic p

- The heart failure never ever cured, except in three conditions:
  - A- Sever anemia
  - B- Hyperthyroidism
  - C- Deficiency of Vit. B_1

if the heart failure occur due to any of the pervious factors, just treatment of causes lead to cure of heart failure.

- The first sign of hypotension is:
  - Dizziness
  - Because of ↓ BF to the brain.

- Why the count of RBCs and Hb is high in fetus and infants until 7th month?
  - Due to intrauterine hypoxia.

★ All the physiological factors that affect the ABP lead to
  - ↑ Systolic pressure

with just two factors cause ↓ diastolic pressure which are:
1- Hot Weather.
2- Exercise.

Lecture 3 Follow of physiological factors affect ABP.

7- Exercise:-

- ABP ↑ 5-20 mmHg than normal.
- ↑ mainly by sympathetic stimulation.
- Normal HR is about 60-100 during rest per minute.
  During exercise HR ≤ 180 beat/minute
  more than 180 beat during exercise mean we have real problem. "Heart failure".
- ECG during exercise: we put the leads of ECG on patient during doing of exercise, and observe the patient ABP. If the ABP decreased we should stop the ECG and exercise, because that is induction of dangerous "may be ischemia or atherosclerosis".

8- Sleep:

- Low ABP, Because the body and brain in rest state.
- High ABP just during nightmares.

9- Emotions:

- Increases the ABP due to sympathetic stimulation.

10- Sexual intercourse:

- ↑ABP due to sympathetic NS.

11- Temprature:

- Hot ↑ ABP due to sympathetic NS.

Regulation of ABP

- Cardiac center: collection of nerve cells inside the CNS
- Site: Medulla oblongata.

Cardiac center devided into

Cardiac inhibitory center

Stimulation of it leads to

Bradycardia

↓Co

↓ABP

Vasomotor center

VCC Vasoconstrictor center

Stimulation

↑discharge of sympathetic impulses

1- BVs → V.C → ↑PR

2- Heart → ↑HR → ↑Co

3- Adrenal Medulla ↑secretion of Adrenalin and noreadrenalin that inforce the pervious two functions.

VDC vasodilator center

Stimulation

1- BVs → VD → ↓PR

Just

* CIC and VDC are stimulate at same time or inhibited together. Which cause
  1- VD of BVs → ↓PR
  2- Heart → ↓HR → ↓Co
  3- inhibit the secretion of Adrenalin and noradrenalin.

The ABP regulated by three mechanisms:

(A) Short-Term regulation mechanism.

(B) Intermediate Regulation mechanism

(C) Long-Term regulation Mechanism.

(A) Short-Term Mechanism:

- Properties:
  - Potent Mechanism "Strong"
  - Rapid Mechanism, occur within seconds
  - Nervous Mechanism.
- Include the following mechanisms:
  - A- Arterial baroreflex.
  - B- Arterial chemoreflex.
  - C- CNS ischemic response
  - D- Abdominal Compression reflex.

(A) Arterial baroreflex:-

- Site: 1-Carotid sinuses
  2-Aortic arch.
- It's the strongest receptors for regulation of ABP.
- Example:

↑ABP → Stimulation of baroreceptors → Send signals by Buffer nerves" Vagus and glossopharyngeal nn." to Cardiac Center → 1- Inhibition of VCC.
2- Stimulation of CIC and VDC

changes → A- Bradycardia → ↓CO
B- V.D of BVs → ↓PR
C- ↓Adrenalin secretion.

★ Vagal tone:

- Definition:- Continuous inhibitory effect on the heart by vagus nerve During Rest.

- Vagus nerve supply the atrial part of heart but not the ventricles.

B Arterial chemoreflex:

- Site: 1- Carotid body
  2- Aortic body
- Just minor effect to CVS, The major effect is to respiratory system.
- Work only When the ABP is below 60 mmHg.
- Example:

↓ABP < 60 → Stimulation of peripheral chemoreceptors → send signals by buffer nerves to Cardiac center → 1- Inhibition of CIC and VDC
2- Stimulation of VCC

Changes → 1- ↑HR → ↑CO
2- V.C of BVs → ↑PR
3- ↑ secretion of adrenalin: "Catacolamine"

C CNS ischemic response:

↓ABP < 60 mmHg → ischemia → Hypoxia → 1- Inhibition of CIC and VDC.
2- Stimulation of VCC.
→ ↑HR, ↑PR, ↑ Catacolamine. → Maintain cerebral blood flow

The brain is the first organ sensitive to hypoxia.
The Heart " " second " " " " .
The kidneys serve " Third organs " " " .

* Cushing reflex:
  ↑ the intracranial pressure → cushing reflex
  → Similar action of CNS ischemic response.

D- Abdominal Copression reflex:
During stimulation of VCC → Stimulation of Abdominal muscles → Contraction of Abd. muscles → ↑ the intrabdominal pressure → Squezzing of the venous blood → ↑ VR to the heart → ↑ CO → ↑ ABP.

B- Intermediate term mechanism:

- Properties:
  - Work to support the short-term mechanism.
  - Occur within minutes to hours.
  - Work when short-term mechanism fatigue but still working.
- Include the following mechanisms:-
  - A- Capillary fluid shift mechanism.
  - B- Stress relaxation mechanism.
  - C- Renin-Angiotensin mechanism
  - D- Right atrium mechanism.

(A) Capillary fluid shift mechanism:

- Mainly to shift the fluid from capillaries to tissue or from tissue to blood.
- This mechanism depend on two pressures:
  1- Hydrostatic pressure HP
  2- Osmotic pressure OP
  And the BV.
- The HP depend on the ABP in its action, for filtration of fluid from capillaries to tissue.

Example:-

First → ↑ BV → ↑ VR → ↑ CO → ↑ ABP → ↑ HP → ↑ Filtration of fluid → excrete of fluid from BVs to tissue → Return the BV to normal → normal ABP.

Second → ↓ BV → ↓ VR → ↓ CO → ↓ ABP → ↓ HP → ↓ Filtration → ↑ OP → ↑ reabsorption of fluid from tissue to blood → ↑ BV to normal → normal ABP.

* Lect 4

(B) Stress relaxation mechanism:-

- It is the weakest mechanism of all mechanisms.

Example:

↑ ABP → Strech the walls of arteries for while → Relaxation of the smooth muscles around the artery → V.D → ↓ ABP.

C- Renin-Angiotensin mechanism:-

*What is the function of Renin?

- Conversion of Angiotensinogen that came from live to Angiotensin I in kidneys, Angiotensin I Convert to Angiotensin II in lungs by ACE "Angiotensin converting enzyme".

*Example:

↓ABP → ↓BF to kidney → Renal ischemia → ↑Renin secretion from Juxtaglumeolar cells → ↑ Angiotensin II →

1- Strong V.C of BVs.

2- ↑ Secretion of aldosterone which lead to → ↑ Absorption of Na to blood. ↑ Water retension to blood → ↑BV → ↑ABP.

- Some drugs for hypertension are "ACEI" Angiotensin Converting enzyme inhibiting → V.D of BVs → ↓ABP.

D Right atrial mechanism:-

- Functions of right atrium:

1- Secretion of ANP.

2- Contain Streech receptors for regulation of ABP.

- Right atrium affected by (BV)

Example:

↑BV → ↑VR → ↑ BV and P inside the right atrium → ↑P >4 mmHg → Stimulation of streech receptors → Send signals to Cardiac center → Changes →

1- Generalized V.D of BV → ↓PR.
2- ↑ secretion of ANP → ↑ excretion of Na and water → ↑ urine volume
3- Inhibition of ADH secretion → ↓ H2o reabsorption → ↓ BV → Normal ABP.

C- Long-Term Mechanism:-

- Properties:
  - Inforce the short and intermediate mechanisms.
  - Slow mechanism occur with days.
- Include the following mechanisms:-
  - A- Glumeular filtration - GF
  - B- Secretion of aldosterone.

Example.

(A)

↓ ABP → ↓ HP → ↓ GF in kidney → ↓ volume of urine → ↑ BV → ↑ ABP.

(B)

↑ ABP → 1- ↑ GF → ↑ urine volume
2- ↓ Renin → ↓ Aldosterone → ↑ Na and H2o in urine → ↑ volume of urine.

NOTES:

* No variable level for:
  1- Blood gases
  2- PH

* Why the diastolic P ↓ during exercise or hot weather?
  Because they lead to V.D of cutaneous BVs.

* Who are the people that live with normal bradycardia e.g HR = 50 l-minute?
  Just the athletics.

* Why the systolic P ↑ due to physiological factors that affect the ABP?
  They lead to ↑HR → ↑ABP, mainly systolic pressure.

* Why we fear from bradycardia rather than tachycardia?
  Because bradycardia mean Heart block.

* Why we concentrate on the diastolic p rather than systolic pressure during ABP measurement?
  Because the systolic pressure is variable and affected by many factors.

* The Parasympathomemetic drugs ↓ABP by ↓HR but not the Co, because no parasympathetic supply to ventricles.
Co depend mainly on the strength of ventricular contraction which supply by Sympathetic nervous sysetem.

* ↑ Blood volume →
Stimulate 1- Capillary fluid shift mechan..
2- Right atrial mechan.

* Renin is an enzyme because it secreted and work on kidney, if it is hormone it should not working in kidney.

* Why the diabeteic patient has continuous diuresis?
Due to ↑ glucose excretion in urine →
↑ osmotic pressure in renal tubules →
Absorption of fluid from blood to kidney → ↑ urine volume.

* The diuretics "Drugs" may → Hypovolemia → Cardia arrest.

* Hypoglycemia and hypotension are more dangerous than Hyper....

ECG

"Electrocardiogram"

- Definition: - Recording of electrical activity of the heart from the surface of the heart.

- Importance: "To know about" diagnosis of certain diseases:
  1- Arrythmia.
  2- Hypertrophy "Atrial or ventricular"
  3- I → I "Myocardial infarction".
  4- Drugs. "Digitalis" → mainly.
  5- Electrolytes disturbance, → mainly K^+
  6- Ischemia.
  7- Pericarditis.

- Digitalis: - Use for inforce the contraction of heart.
  - Described for patient with below 35 ejection friction of the heart
  - Normal ejection friction is about ≥ 65.

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* Recording points on the body surface:
  There are nine points devided as:
  (A) Six points on the chest surface:
- V_1 → V = Mean Voltage.
  Located in the 4th intercostal space, just to the right border of Sternum.

- V2 → Located in the 4th intercostal space just to the left border of Sternum.
- V3 → Located inbetween the V2 and V4.
- V4 → in the 5th intercostal space at the level of the left mid-clavicular line.
- V5 → in the 5th intercostal space at the level of the left anterior axillary line.
- V6 → In the 5th intercostal space at the level of the left mid-axillary line.

(B)- Three points in the limbs.

- aVR a = Augmented
  V = Voltage
  R = right arm

- aVL Left arm.
- aVF Left foot.

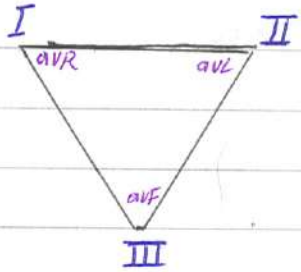
The right cathod of foot located on the floor for charges.

★ ECG Leads:-

There are 12 leads, for nine points of ecg.

(A) Unipolor leads:
For the chest and limbs.

(B) Bipolor leads:
I , II , III



* Lead I : difference effort between left arm and right arm.

* Lead II : Between right arm and left leg.

* Lead III : Between left arm and left leg.

Lecture "5"

- The ECG consist of Waves, segments and intervals.

A-Waves

- There are five waves appear normally:
P - Q - R - S and T wave.

For each wave we should talk about "Direction(A) - Cause(B) - Abnormalities(C)".

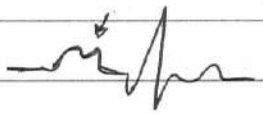
1- P wave:

(A) - It's + ve wave "e.g it' directed upward

(B) - Related to Atrial depolarization. "Right and left atria".

C- Abnormalities of P wave:

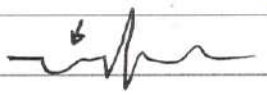
1- P Mitral.
2- P pulmonale.
3- Disappeared P wave.
4- Inverted P wave

1- Mitral P wave: 

- Appear due to:
  Left atrial hypertrophy
- Changes:
  Large, notched with long duration because the hypertrophic muscles cause delay of conduction.

2- Pulmonale P wave:

- Appear due to:
  Right atrial hypertrophy.
- Changes:
  Tall and peaked wave with long duration.

3- Inverted P wave:  -ve wave.

- Appear during
  1- AV nodal rhythm.
  2- Dextrocardia.
  3- تركيب أسباب [illegible]
- Changes:
  -ve P wave, directed downward.

*AV nodal rhythm mean the AV node act as the pacemaker if the SA pacemaker stoped.

4-Disappeared P wave:

- Appear due to :-
  1- Atrial fibrillation.
  2- Severe hyperkalemia.
- Changes:
  No P wave at all.

2- QRS complex:

Related to ventricular depolarization

Q wave
A- -ve wave
B- Depolarization of interventricular septum.

R wave
A- +ve wave
B- Depolarization of apex of heart and ventricular wall.

S wave
A- -ve wave
B- Depolarization of last two areas in the heart
- Left ventricle: • Posterobasal Part.
- Right ventricle: • Pulmonary conus.

C- Abnormalities:-

We can see abnormalities on QRS complex during four disease:

1- Ventricular hypertrophy
2- Bundle branch block.
3- I — II
4- Electrolytes disturbances
Mainly hyperkalemia → Prolongation of QRS complex.

- In ECG

Note: * We can't see the repolorization waves of atria because the thickness of ventricular muscles is more than atria.

* The amplitude of QRS complex is higher than P wave due to thickness of muscle fibers. But the duration of P wave is longer than QRS complex due to few no. of gab junction in VA node that located in the atrium.

3 T wave;

A • +ve wave.

B • Ventricular repolorization.

C. Abnormalities:

1-Inverted T wave

• Due to:

1-Severe ventricular hypertrophy
2-Ischemia.
3-Bundle branch block.
4-Digitalis toxicity

2-Elevated T wave

Due to:

1- Hyperkalemia Physiolically
2- Exercise or Sympathetic stimulation.
3- Fear

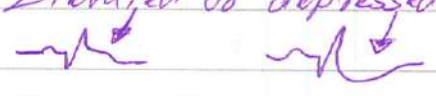
B-Segments

• Part of ECG recorded on the isoelectric line between two waves "End of wave + start of wave"

• We will disscuse S-T segment just because it is the most important segment.

* S-T segment:
  • Started from the end of S wave and ended in the begining of T wave.

  • This segment is very important espcially for diagnosis of I~II, according to elevation or depression of this segment.
  Also to be sure of I~II dignosis we should test the cardiac enzymes.

  • 3 important marks of I~II:
    1- ischemia → inverted T wave
    2- Injury → Elevated or depressed S-T segment 
    3- Necrosis → Deep Q wave in V_1, V_2, V_3 and V_4

• Levels of I~II in ECG: "Marks in ECG".
A- First hours:-
Elevation or inverted S-T segment.

B- After 2 or 3 days of treatment:
Inverted T wave, and deep Q wave.

C- After weeks:
- The S-T segment, return to normal.

D- After monthes:
- The T wave became normal.
- But the Q wave stell deep due to necrosis of ~~the~~ cardiac muscles.

* Some times Patient with I~~—~~II came with typical sings of I~~—~~II, but he looks normal in the ECG, so we should test the cardiac enzymes to be sure.
* Any patient with I~~—~~II, should take a rest.

* The R wave stell normal during I~~—~~II with normal amplitude, but if the I~~—~~II is severe we observe poor R wave which mean "Heart failure".

* Types of I~~—~~II:

There are four ~~types~~ of I~~—~~II

A- Anterior I~~—~~II
B- Lateral I~~—~~II
B- Posterior I~~—~~II
C- Inferior I~~—~~II

A- Anterior MI:

- Sings:-
  1- Tachycardia.
  2- Retrosternal pain
- ECG "V1, V2, V3 and V4":-
  1- Elevated or depressed S-T segment.
  2- Inverted T wave.
  3- Deep Q wave.

The R wave is normall in V1. -ve R wave

B- Posterior MI:

- Sings:-
  1- Tachycardia.
  2- Retrosternal pain
- ECG "V1, V2":-
  1- Elevated or depressed S-T segment.
  2- Inverted T wave.
  3- Deep Q wave.
  4- +ve R wave in V1

C- Inferior MI:-

- Signs:
  1- Bradychardia
  2- Epigastric pain
- ECG: "V1, V2, V3, and V4"
  1- Elevated or depressed S-T segment.
  2- Inverted T wave.
  3- Deep Q Wave.

Axis Deviation :-

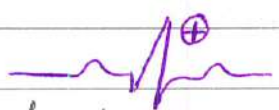
- Normally the axis of heart is located between (-30)° – (+110)° Directed downward to the left.

- The axis of the heart may changes ~~by~~ during some disease which cause Right or left axis deviation.

- How we can diagnose the right or left axis deviation by ECG ?
  Mainly by lead I and lead III

Examplex:

(A) Normally :-



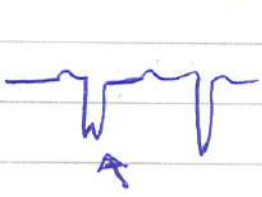
Lead I

Lead III

(B) Left axis deviation :

(C) Right axis deviation :

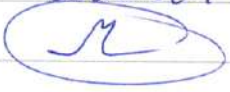
Some times in certain cases the right or left axis deviation appear like:



- if it -ve in lead I that is, mean right axis deviation with right bundle branch block.
- if it -ve in lead III, mean left axis deviation with left bundle branch block.
  Called Double R wave.

- Another way to determine the right or left bundle branch block:

- Right
  V1, V2
  The R-wave is doubled.



- Left
  V5, V6
  The R-wave is doubled.

* Causes of left axis deviation:-
  1- Left ventricular hypertrophy.
  2- Left bundle branch block.
  3- Short and Obese people as well as pregnant women but normal "Physiological"

* Cause of Right axis deviation:
  1- Right ventricular hypertrophy
  2- Right bundle branch block
  3- Tall and thin people "physiologically".

C. Intervals:

- Part of ECG, consist of segment and one or more waves.

The most important interval is P-R interval.

* P-R interval:
- Started from the begining of P wave, ended in the begning of QRS complex.

- Appear due to:
  AV nodal delay.

The duration of AV nodal delay is about 0.1 second which it is very important for collection of blood into the ventricles befor ventricular contraction.

Abnormalities of P-R interval:

A- Prolongation of P-R interval:

very long duration during:

1- First degree of heart block.

2- Increase the activity of parasympathetic NS. by vagal tone.

B- Shortining of P-R interval:-

very short duration during:-

1- AV-nodal rhythm.

2- Increase the activity of sympathetic NS.

3- WPW syndrome.

'Ventricular hypertrophy'

A- Right ventricular hypertrophy:-

1- very long + R wave → V1 V2

2- very deep S wave → V5 V6

B- Left ventricular hypertrophy:-

1- Very deep S wave → V1 and V2

2- very long - R wave → V5 V6

Notes:-

* U wave appear just during **Hypokalemia**.

* Marks of Hyperkalemia:
  1- Tall and deep T wave.
  2- Absence of P wave.
  3- Prolongation of QRS Complex.

* Marks of Dextrocardia:
  1- Inverted P wave.
  2- +ve R wave in V1.
  3- Right axis deviation.

* Marks of WPW syndrome:
  1- +ve R wave in V1
  2- Shortining of P-R interval

* Causes of +ve R wave in V1:
  1- Posterior I—II
  2- Right ventricular hypertrophy.
  3- Right bundle branch block.
  4- WPW syndrome.
  5- Dextrocardia.

Lecture 6 "Heart rate"

HR is: The left ventricular beats.

* Physiological factors that affect the HR:

1- Age:

- Newly borns:
  High HR because absence of vagal tone
- Adults:
  Lower HR due to stronger vagal tone.

2- Sex:

- Female: Higher HR
- Male: Lower HR.

3- Circadian rhythm:

- Morning: Low HR
- Evening: High HR.

4- Rest and sleep:

Lower HR due to less activity of sympathetic NVS.

5- Physical training:

- HR is low → About 50 / minute.
- Just in the athletics because of
  - A - ↑ vagal tone.
  - B - Left ventricular hypertrophy.

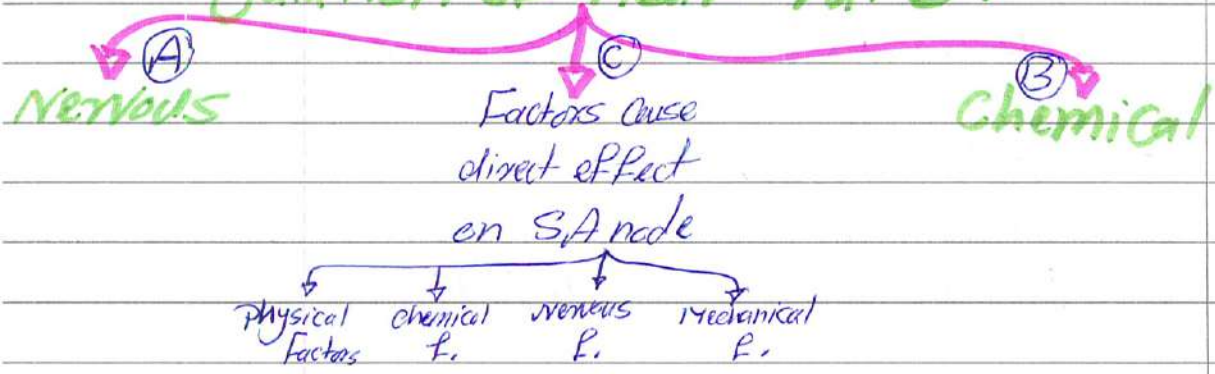
6- Posture:

- Sudden Standing up lead to ↑ HR due to Marry's law "see later".

- The normal heart rate is about:
  60 - 100 / minute
  ↑ HR > 100 → Tachycardia
  ↓ HR < 60 → Bradycardia.

- Sudden standing up cause tachycardia due to ~~inc~~ ↓ of ABP. ↓ABP is due to directed of blood downward ⟹ ↓VR → ↓ABP → ↑HR.

• Regulation of heart rate:



A- Nervous Mechanism :-

- Mainly by three mechanisms:

1- Supraspinal "Higher" center of brain.
2- Reflexes from CVS.
3- Reflexes from extravascular structures.

1 Supraspinal "Higher" center of brain:

- Mainly by: A- Cerebral cortex.
  B- Hypothalamus.
  C- Limbic system.
  D- Respiratory center.

- First by A- Cerebral cortex.
  B- Hypothalamus.
  C- Limbic system.

- Most of times the pervious three structure lead to ↑ HR due to Emotions "- shy
  - Happiness.
  - Love.

- But some emotions lead to ↓HR as: shocking news.
  → ↓HR → ↓PR → ↓ABP → Cardiac arrest.

- Also there is "Conditioned reflex." by hearing good news → ↑ HR. by intact the cerebral cortex.

- Second: by: D- Respiratory center
  "Know as Respiratory Sinus arrhythmia"
  - Inspiration → ↑HR
  - Expiration → ↓HR
  Especially in children.

- How these occurs?
  ↑HR is due to: 1- Bainbridge reflex
  ↑ RAP – ↑ HR

- During inspiration the plura and lungs inflated which lead to ↓ the intra-thoracic pressure → help the VR → ↑ RAP → ↑ HR

[2-] Irradation of impulses from respiratory center to cardiac center:

Stimulation of Apneustic center → sen signals to dorsal or Ineurons in medulla oblongata → signals stimulation to VCC of cardiac center → ↑HR.

[3]- Lung inflation.

By stimulation of pulmonary strech reflex.

2 Reflexes from CVS:

- The most important mechanism by:
  - A- Marey's law or reflex.
  - B- Bainbridge reflex.
  - C- Left ventricular reflex.
  - D- Coronary chemoreflex.

A- Marey's law:

- Said: ↑ABP → ↓HR
  ↓ABP → ↑HR

Just ✓✓

XXXnot ↑HR → ↓ABP or ↓HR → ↑ABP XXX

- This law depend on four action:

1- Arterial baroreceptors mainly.

↑ABP → Stimulation of arterial baroreceptor → send signals by buffer nerves → Inhibition of VCC and Stimulation of CIC and VDC which cause

1- V.D
2- Hypotention
3- Bradycardia.

2- Inhibition of respiratory center
3- Inhibition of ADH secretion.
4- Reduce the activity of brain.

B- Bainbridge reflex:

↑RAP → ↑HR.

- Mainly by stimulation of atrial baroreceptors which cause:
  1- V.D
  2- Hypotension
  3- Tachycardia ←

- ↑RAP lead to six changes:
  1- Tachycardia
  2- Tachypnea
  3- Hypotention due to V.D of BVs.
  4- Stimulation of ANP secretion
  5- Inhibition of ADH secretion
  6- V.D of coronary BVs.

C- Left ventricular Strech reflex:

- Distension of left ventricle → stimulation of ventricular baroreceptors which cause:
  1- Hypotension
  2- Bradycardia
  3- V.D.

D- Coronary chemoreflex:

- Injection of Sertonin in the coronary BVs cause stimulation of coronary chemoreflex which cause:
  1- Apnea.
  2- Hyperphea.
  3- Hypotension.
  4- ~~Tachy~~ Bradycardia.

- Some cases of MI lead to necrosis → Release of certain substances include Sertonin → Stimulation of coronary chemoreflex.

- Notes:-

Any Stimulation of Atrial or pulmonary baroreceptors lead to: 1- V.D.
2- Hypotension.
3- Tachycardia.

Any Stimulation of Arterial or ventricular baroreceptors lead to: 1- V.D
2- Hypotension
3- Bradycardia

Lecture 7 Follow of nervous regulation of HR.

3- Reflexes from extravascular Structures:-

Five organs:

1- Respiratory system.
2- Skin
3- Eyes
4- Sk. muscles
5- Trigger zone or area.

1- Respiratory system by two reflexes:

A- Pulmonary chemoreflex:

- Injection of Sertonin to the pulmonary BVs. lead to Stimulation of pulmonary chemoreflex which cause: 1- Apnea.
2- Hyperpnea
3- Hypotension
4- Bradycardia

B- Pulmonary baroreceptors "Strech reflex":-

- Stimulation of pulmonary baroreceptors by lung inflation cause 1- VD
2- Hypotension
3- Tachycardia.

2- Skin:

A. Cold, Hot and mild pain lead to ↑ HR mainly by Hypothalamus.

B. Severe pain "I" lead to ↓ HR by Stimulation of cerebral cortex.

3- Sk. muscles: "Alarm-smirk reflex"

Contraction of Sk. muscles lead to:

1- Tachycardia = ↑HR

2- Tachypnea.

4- Eyes "Oculo-cardiac reflex":

Applying Pressure to eyeball → Stimulation of the receptors in the eye → Send signals by oculo-motor nerve to cardiac center → Stimulation of CIC → ↓ HR.

5- Trigger Zone or area:

- Very sensitive areas:
  1- Larynx
  2- Epigastric region
  3- Testis

Apply pressure on any of them → ↓ HR.

B-Chemical regulation :-

Mainly by:-

1- Gases

2- Hormones, drugs and chemicals.

1- Gases: A- O_2 B- Co_2

A- O_2

1- Moderate hypoxia: ↑HR

by three mechanism:

A- Direct mechanism:

By Stimullation of SA node.

B- Central mechanism:

By inhibition of CIC:

C- Reflex mechanism:

By Stimulation of peripheral chemoreceptors → signals from buffer nerves → Stimulation of VCC.

2- Severe hypoxia: ↓HR

by two mechanism:

A- Direct inhibition of SA node.

B- Paralysis of Cardiac center.

B- Co_2:

1- Moderate hypercapnia: ↑HR.

By three mechanism:

A- Inhibition of CIC → ↑HR

B- Stimulation of VCC by peripheral chemoreceptors.

C- Stimulation of VCC by central chemoreceptors

2- Severe hypercapnia: ↓HR

By ~~three~~ two mechanisms:

A- Direct inhibition of SA node.

B- Paralysis of Cardiac Center.

2- Hormones, drugs and chemicals:

1-Adrenaline
2-Noradrenaline
3-Thyroxine
4- ATropin
5-Histamine
6- Bile salt.

1- Adrenaline:

A- Large does: ↓HR

V.C of BVs → ↑PR → ↑ABP → ↓HR

According to Marey's law.

B- Small does: ↑HR

By direct stimulation of SA node.

2-Noradrenaline: ↓HR

Strong V.C of BVs → ↑PR → ↑ABP → ↓HR

According to Marey's law.

3- Thyroxin: ↑HR

By: 1- ↑ Metabolism

2-Direct effect on SA node.

4- Histamin: ↑HR

V.D of BVs → ↓PR → ↓ABP → ↑HR

According to Marey's law

5- Atropin: ↑HR "Block of parasymathetic"

By inhibition of Parasympathetic NS → Decrease vagal tone → ↑HR.

6- Bile salt: ↓ HR

↑ Bile salt during obstrective Jaundice due to gall stone → ↓ HR by:

1- Direct inhibition of SA node.

2- Stimulation of CIC.

Notes:-

* Oculomotor nerve is:

Third cranial nerve consist of two parts: 1- Somatic for the eye muscles

2- Parasympathetic for:

A- Torsal muscles.

B- ciliary muscles

* Signs of Obsterective Jaundice:

1- Severe Jaundice.

2- Bradycardia.

3- Itching.

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"Cardiac Output"

- Mean: The volume of blood that ejected from each ventricle per minute.
  About 5,5 L/min during rest.
  $Co = SV \times HR$

- SV Mean: The volume of blood ejected by each ventricle per beat.

- VR = Cardiac input.
- The volume of blood ejected from left ventricle is similar to volume of blood ejected from right ventricle.

- $SV = EDV - ESV$.
  - Systole Mean:
    Ejection of blood from left and right ventricles.
  - Diastole Mean:-
    Ventricular filling of blood.

- EDV:
  - it is the amount of blood in the ventricle at the end of diastole.
  - it is about 130 mL/beat
  - The ventricles during rest should be full of blood about 130 mL, Once the systole occur just 80 mL ejected and 50 mL stell as ESV.

• ESV:
The volume of blood stell in the ventricle after systole.

* Ejection fraction:

$$= \frac{SV}{EDV} \times 100$$

$$EF = \frac{80}{130} \times 100 = \boxed{61}$$

• The ejection fraction of heart range from
60 - 65
Recorded by Echocardiogram..

• Decrease the contractility of ventricles lead to Increase the ESV

• Increase the force of ventricular contraction lead to Decrease the ESV.

• During mitral stenosis the EDV will be less than 130 mL.

• During mitral regurgitation part of the ejected blood pass to atria the ethe part pass to the vessels.

* Factors affect the EDV:

1- RAP.
2- BV.
3- Venous tone.
4- Intrapericardial P.
5- Atrial fibrillation
6- Intrathoracic P.
7- Sk. muscle contraction
8- Posture
9- Ventricular compliance and stiffness.

1- RAP: ↑EDV
↑VR → ↑RAP → ↑EDV.

2- BV:
- ↓BV → ↓VR → ↓EDV
- ↑BV → ↑VR → ↑RAP → ↑EDV

3- Venous tone or Venoconstriction:
- Venous constriction → ↑VR → ↑RAP → ↑EDV

4- Intrapericardial P:
↑ The intrapericardial P → ↑Compression of ventricle → ↓EDV.

5- Atrial fibrillation:
(350 - 400 systole of atria per minute)
↓EDV, due to fast but weak contraction of atria.

6- Intrathoracic P.:

During inspiration

Reduce the intrapleural pressure → ↓ The intra-thoracic pressure → help VR to the heart → ↑RAP → ↑ EDV.

7. Sk. muscles Contraction:

Contraction of Sk. muscles → ↑VR → ↑RAP → ↑EDV.

- Very important mechanism during sudden Standing up.

8- Posture:

Sudden Standing up → ↓EDV due to ↓VR.

9. Ventricular:

A- Compliance

↑ Compliance of ventricle → ↑EDV

B- Stiffness:

↑ Stiffness of ventricle → ↓EDV

Lect. 8 "Last lecture" Follow of CO

Factors affect CO:

(A) Factors affect HR

(B) Factors affect SV

A-Factors affect HR:

- The normal HR during rest rang from 60-100 /minute.

(A) Decrease the HR:

1- From 70-50 /minute, will not affect the Co. e.g the Co will be normal.

2- Below than 50/minute, will cause ↓ of Co because the SV will not be equal to the HR.

(B) Increase the HR:

1- ↑ HR to 180 /min the ▦ Co will ↑ during exercise due to sympathetic stimulation which cause strong ventricular contraction.

2- ↑ HR to 170 or more during rest The Co will ↓ or constant, because there is no sympathetic stimulation.

3- ↑ HR > 200/min → ↓ SV → ↓ CO.

B-Factors affect SV:

1- Cardiac pumping power
2- Venous return.

1- Cardiac Pumping power:

- Mean the maximum Co that pumped by each ventricles per minute.
- Affected by: A-Preload
  B-Afterload.
  C-Sympathetic nrs.
  D-Ventricular hypertrophy.

A-Preload: "Starling law" ↑Co

- Mean EDV.
  ↑ length of muscle → Stronger contraction within limits.
- Maximum Co during preload is about 10 L/min

B-Afterload: "Aortic resistance" ↓Co

Lead to ↓Co due to:
1- Hypertension "Mainly"
2- Polycythemia.

C- Sympathetic Stimulation

1- Minimal Stimulation:
↑Co to about 15 ml/min
2- Maximum Stimulation:
↑Co to about 25 L/min

D- Ventricular hypertrophy:
Normally seen in the athletics:
↑Co to about 35 L/min

2- Venous return:- " Cardiac input "
Affected by: A-RAP
B-MCP
C-Resistance to the VR.

A- RAP:
↑RAP → ↓VR → ↓Co
- Due to high resistance of right atrium to the VR due to ↑ the RAB.

B-MCP. Mean circulatory pressure.
- Depend on BV.
1- Hypovolemia → ↓MCP → ↓VR
2- Hypervolemia → ↑MCP → ↑VR.

During Vena Constriction → ↑MCP → ↑VR → ↑Co

C-Resistance of the VR:
- Majorly in veins.
- Minorly in arterioles

* Any volume enter the heart should exert with same volume from the heart.

Regulation of CO :

(A) Extrinsic Mechanism

(B) Intrinsic Mechanism.

A- Extrinsic Mechanism :-

Mainly by : HR and SV.

1- ANS

2- Hormones and drugs

1- ANS : A- Sympathetic :

its stimulation lead to :

1- Increase the force of contraction.

2- ↑ HR

3- ↑ Coronary BF

⟶ ↑ CO

B- Parasympathetic :

its stimulation lead to :

1- ↓ Force of contraction

2- ↓ HR

3- ↓ Coronary BF

⟶ ↓ CO.

2- Hormones and drugs:

1- Digitalis
2- Xanthines

↑Co due to ↑ the force of contraction.

3- Catacolamines:

- ↑ mainly ↑ HR
- ↑Co by ↑ force of contraction.

4- Thyroxin:

1st → ↑HR
2nd → Force of contraction → ↑Co

5- Glucagon:

↑ Force of contractility → ↑Co

6- Acetylcholine:

↓ HR mainly and ↓ force of contraction
→ ↓ Co

7- Hypoxia, Hyperkalemia and acidosis, ischemia
↓ force of contractility → ↓ Co.

B- Intrinsic Mechanism:

By: For SV only

1- Heterometric Autoregulation.
2- Homeometric Autoregulation.

1- Heterometric Autoregulation

- Mainly by change of muscle length of cardic muscles.

"Mainly by Starling law"

Stretch of cardic muscle → Stroger contraction → ↑SV → ↑Co

2- Homeometric Autoregulation

- Without changing of muscle fiber length.

Physiological factors affect the Co:

1- Sleep:

Normal↓, because the HR is low as well as the SV.

2- Exercise: ↑Co

3- Emotions ↑Co

4- Pregnancy:

↑Co due to ↑uterine BF.

5- After meal: ↑Co due to ↑VR from high BF to stomach.

6- Posture:

Sudden sitting down → ↑co due to absence of gravity.

but sudden standing up → ↓Co due to ↓VR
to heart → ↓Co.

7-Temperature:
A-Hot ↑Co Due to:
1- VD of cutaneous BVs
2- ↑ HR.
B- Cold ↑ Co due to;
الارتجاف

• Notes :—
Cardiac cycle mean one systole and diastole.
• Systole represent → 0,3 sec
• Diastole represent → 0,5 sec